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Task Force 4: Systemic Hypertension

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General Considerations

Systemic hypertension is the most common cardiovascular condition observed in competitive athletes. The diagnosis of hypertension is based on the presence of blood pressure persistently at or above certain levels as measured by routine sphygmomanometry on at least three separate occasions (Table 1). In determining the level of competitive athletic activity that a hypertensive person may assume, it is also important to ascertain the degree of hypertension-related target organ damage (1). Although hypertension is associated with an increased risk for sudden death and complex ventricular arrhythmias (2), this disease has not yet been incriminated as a cause of sudden cardiac death in young competitive athletes

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Table 1. Classification of Hypertension by Age in Children and Adolescents

	Magnitude of Hypertension*			
	Mild (stage 1) (mm Hg)	Moderate (stage 2) (mm Hg)	Severe (stage 3) (mm Hg)	Very Severe (stage 4) (mm Hg)
Children				
6–9 yr old†				
Systolic BP	120–124	125–129	130–139	≥140
Diastolic BP	75–79	80–84	85–89	≥90
10–12 yr old†				
Systolic BP	125–129	130–134	135–144	≥145
Diastolic BP	80–84	85–89	90–94	≥95
Adolescents				
13–15 yr old†				
Systolic BP	135–139	140–149	150–159	≥160
Diastolic BP	85–89	90–94	95–99	≥100
16–18 yr old†				
Systolic BP	140–149	150–159	160–179	≥180
Diastolic BP	90–94	95–99	100–109	≥110
Adults				
>18 yr old‡				
Systolic BP	140–159	160–179	180–209	≥210
Diastolic BP	90–99	100–109	110–119	≥120

*Applies to patients who are not taking antihypertensive drugs and are not acutely ill. When the systolic and diastolic blood pressures (BP) fall into different categories, the higher category should be selected to classify that patient's blood pressure status. In adults, isolated systolic hypertension is defined as a systolic blood pressure ≥140 mm Hg and a diastolic blood pressure <90 mm Hg and staged appropriately. Blood pressure values are based on the average of three or more readings taken at each of two or more visits after the initial screening. †Adapted from the recommendations of the Second Task Force on Blood Pressure Control in Children (Pediatrics 1987;79:1–25) to be consistent with the classification in adults. ‡From the Fifth Report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure (JNC V). Arch Intern Med 1993;153:154–83.

(3,4). Consequently, most of the following considerations pertain to the effects of training and competition on the progression of hypertension.

Effects of exercise. The possibility exists that the immediate pressor effect of strenuous static activity may be harmful and is based largely on a few reports (5) of cerebrovascular accidents during maximal effort. However, there is increasing evidence that pure static (6) or combined static and dynamic activity, as provided by circuit training (7), may in fact produce a long-term antihypertensive effect. African-Americans may have a greater pressor response during dynamic exercise than non-blacks (8,9). However, there are no data to implicate a less long-term benefit or increased cardiovascular risk from exercise in this population.

In contrast, an impressive body of data (10,11) is available that documents an antihypertensive effect from repetitive dynamic physical activity. Although multiple mechanisms are probably responsible (12), it is worth emphasizing that exercise may normalize blood pressure in some people with mildly elevated levels (13).

All those with hypertension who wish to engage in competitive athletics should increase activity levels gradually so as to avoid the cardiac catastrophes seen in sedentary people who undertake major bursts of strenuous exercise (14). Because the risk of myocardial infarction per unit time appears to be modestly increased during intense exercise, even in those who are well conditioned (14), an exercise test may be appropriate for hypertensive athletes with other coronary risk factors who are >35 years old.

Untreated hypertension in athletes may be accompanied by some limitation in exercise performance (15). Before initiating drug therapy, athletes should be strongly encouraged to adopt healthy life-style behavior and avoid the following: tobacco in any form, excess alcohol, drugs of abuse (especially cocaine), androgens, growth hormones and high sodium intake. The use of antihypertensive drugs may further limit exercise capacity, more so with beta-adrenergic receptor blocking agents than with vasodilators (alpha-adrenergic blocking agents, angiotensin-converting enzyme inhibitors or calcium channel blocking agents) (16). However, performance may be enhanced by reduction of elevated blood pressure and curtailment of exercise-induced hypertensive responses with well tolerated antihypertensive medications. It is advised that the use of diuretic drugs and beta-blockers by competitive athletes has been prohibited by some athletic governing bodies.

Evaluation. Blood pressure should be accurately measured in all who wish to compete in competitive athletics, preferably *before* they begin training. Blood pressure should be measured by routine sphygmomanometry, using the guidelines listed in Table 2 (17). Caution is needed to avoid "white-coat" elevations induced by the anxiety of the examination, particularly in a young person concerned about the examination. If feasible, additional blood pressure recordings outside the office could be obtained in those with high office readings, either with readily available inexpensive home self-recorders (after the accuracy of their use has been validated) or with less accessible and more expensive automatic ambulatory monitors.

Considerable anxiety and, thereby, further elevations in blood pressure may follow the initial recognition of an elevated blood pressure. Therefore, unless the blood pressure is severely elevated (Table 1), the subject should be advised that the initial reading is somewhat elevated but may decrease on repeated measurements and that there is no reason to be concerned until more measurements are taken.

Those with any degree of persistent hypertension should have a thorough history and physical examination (17) and limited laboratory testing to evaluate secondary causes and for target organ damage. The laboratory testing for most subjects observed to have mild to moderate (stages 1 and 2) hypertension should be limited to an automated blood chemistry (glucose, creatinine, electrolytes, cholesterol), hematocrit, urine analysis and an electrocardiogram. If the results of these are abnormal, or if features suggestive of secondary causes are noted by history or physical examination (17), the subject should be referred for additional study and therapy.

Two additional studies, echocardiography and exercise-stress testing, may be useful. However, because of the expense

Table 2. Guidelines for Measurement of Blood Pressure

Posture
Blood pressure obtained in the sitting position is recommended. The subject should sit quietly for 5 min, with the back supported and the arm supported at the level of the heart, before recording blood pressure
Circumstances
No caffeine during the hour preceding the reading
No smoking during the 30 min preceding the reading
A quiet, warm setting
Equipment
Cuff size
The bladder should encircle and cover two-thirds of the length of the arm; if it does not, place the bladder over the brachial artery. If bladder is too short, misleadingly high readings may result
Manometer
Aneroid gauges should be calibrated every 6 mo against a mercury manometer
Technique
No. of readings
On each occasion, take at least two readings, separated by as much time as is practical. If readings vary by >5 mm Hg, take additional readings until two consecutive readings are close
If the initial values are elevated, obtain two other sets of readings at least 1 week apart
Initially, take pressure in both arms; if the pressures differ, use the arm with the higher pressure
If the arm pressure is elevated, take the pressure in one leg (particularly in patients <30 yr old)
Performance
Inflate the bladder quickly to a pressure 20 mm Hg above the systolic pressure, as recognized by disappearance of the radial pulse
Deflate the bladder 3 mm Hg/s
Record the Korotkoff phase V (disappearance), except in children, in whom use of phase IV (muffling) may be preferable when disappearance of the sounds is not perceived.
If the Korotkoff sounds are weak, have the patient raise the arm, open and close the hand 5 to 10 times and then reinflate the bladder quickly
Recordings
Blood pressure, patient position and arm and cuff size

and the absence of data documenting that they provide either diagnostic or prognostic information *about the eligibility for athletic competition* in relation to hypertension, they are not required for routine use. In those subjects in whom an exercise test is performed for other reasons, a marked increase in systolic blood pressure (>240 mm Hg) may presage the development of hypertension under basal conditions and indicate the need for further investigation and antihypertensive therapy. An increase in diastolic pressure is generally evidence for increased peripheral resistance. If an echocardiogram is performed, the presence of significant concentric hypertrophy is an important risk factor in adults (18) and may indicate the need for antihypertensive therapy, even at blood pressure levels that might not otherwise be high enough to mandate therapy.

Types of Hypertension

Because ~95% of all systemic hypertension is primary (essential), we have made no distinction in this report between primary and secondary etiologies of increased blood pressure. Obviously, if secondary forms of hypertension are identified, these should be appropriately managed.

Recommendations

1. The presence of mild to moderate (stages 1 and 2) hypertension (as defined in Table 1) in the absence of target organ damage or concomitant heart disease should not limit the eligibility for any competitive sports. Because there are in fact documented benefits from moderate dynamic and static exercise in lowering the blood pressure and improving other coronary risk factors (1), a persuasive argument can be made for hypertensive subjects to engage in regular forms of exercise. Once having begun a training program, the hypertensive athlete should have blood pressure remeasured every 2 to 4 months or more frequently, if indicated, to monitor the impact of exercise.

2. Athletes with severe hypertension (stages 3 and 4) should be restricted, particularly from high static sports (classes IIIA to IIIC [see Table 1 in Classification of Sports]), until their hypertension is controlled by either life-style modification or drug therapy (and in the absence of evidence of target organ damage). Because competitive athletics may demand more strenuous exercise than is needed for cardiovascular conditioning, these restrictions are appropriate for those with relatively severe hypertension, particularly if target organ damage is present. Despite the apparent logic of this position, it should be emphasized that there are few data documenting that strenuous dynamic exercise among even the most severely hypertensive subjects enhances risk for sudden cardiac death or progression of their hypertensive disease (3,4,19).

3. When hypertension coexists with other cardiovascular diseases, eligibility for participation in competitive athletics is usually based on the type and severity of the other associated conditions.

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Task Force 5: Coronary Artery Disease

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Atherosclerotic Coronary Artery Disease

General Considerations

The most frequent cause of exercise-related cardiac events and sudden death in adults is atherosclerotic coronary artery disease (1). Furthermore, the incidence of myocardial infarction (2,3), cardiac arrest (4) and sudden cardiac death (1) increases transiently during vigorous physical activity. In some studies, a significant proportion of adult victims of exercise-related sudden death had known coronary artery disease (1). Therefore, it is likely that the risk of exercise-related cardiac events in patients with previously diagnosed coronary artery disease is higher than that in apparently healthy subjects.

Necropsy examination of adult victims of exercise-related sudden death usually reveals either advanced coronary artery stenosis or an acute coronary artery lesion, or both (5,6). This observation suggests that exercise-related sudden death can result from either exercise-induced myocardial ischemia in the presence of significant atherosclerotic coronary narrowing or from the progression of a lesion in a diseased coronary artery.

There has been considerable progress in our understanding of coronary artery disease since the previous task force report (7). The previous report considered as significant only those lesions producing >50% narrowing of the lumen diameter. Such lesions are regarded as "hemodynamically significant" because they often restrict coronary artery flow during periods of increased myocardial oxygen demand. It is now appreciated that the morphology of less severe atherosclerotic lesions can be rapidly altered by plaque rupture and thrombosis, leading to unstable angina, myocardial infarction and death (8,9). Acute plaque rupture or thrombosis is present in a high proportion of sudden cardiac deaths in the general population (9) and has also been documented in victims of exercise-related cardiac events (5,10,11). Consequently, such hemodynamically "non-significant" stenoses may increase the cardiac risk of exercise but frequently would not be suspected by presently available